EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	3253	allopurinol	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:08
L2	70314	hypertension	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:09
L3	69227	"blood pressure"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:09
L4	733	L1 and L2	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:09
L5	458	L1 and L3	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:10
L6	210	L4 and @ad<"20020628"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:11
L7	168	L5 and @ad<"20020628"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:11
L8	132	L6 NOT L7	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:43
L9	760	"xanthine oxidase inhibitor"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:55
L10	760	L9	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:55
L11	295	L2 and L9	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:55

EAST Search History

L12	86	L11 and @ad<"20020628"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 11:56
L13	120	L3 and L9	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 12:08
L14	34	L13 and @ad<"20020628"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2008/01/31 12:08

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NEWS 3	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 4	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
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NEWS 10	SEP 13	FORIS renamed to SOFIS
NEWS 11	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 12	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 13	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 14	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 15	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 16	OCT 19	BEILSTEIN updated with new compounds
NEWS 17	NOV 15	Derwent Indian patent publication number format enhanced
NEWS 18	NOV 19	WPIX enhanced with XML display format
NEWS 19	NOV 30	ICSD reloaded with enhancements
NEWS 20	DEC 04	LINPADOCDB now available on STN
NEWS 21	DEC 14	BEILSTEIN pricing structure to change
NEWS 22	DEC 17	USPATOLD added to additional database clusters
NEWS 23	DEC 17	IMSDRUGCONF removed from database clusters and STN
NEWS 24	DEC 17	DGENE now includes more than 10 million sequences
NEWS 25	DEC 17	TOXCENTER enhanced with 2008 MeSH vocabulary in MEDLINE segment
NEWS 26	DEC 17	MEDLINE and LMEDLINE updated with 2008 MeSH vocabulary
NEWS 27	DEC 17	CA/CAplus enhanced with new custom IPC display formats
NEWS 28	DEC 17	STN Viewer enhanced with full-text patent content from USPATOLD
NEWS 29	JAN 02	STN pricing information for 2008 now available
NEWS 30	JAN 16	CAS patent coverage enhanced to include exemplified prophetic substances
NEWS 31	JAN 28	USPATFULL, USPAT2, and USPATOLD enhanced with new custom IPC display formats
NEWS 32	JAN 28	MARPAT searching enhanced
NEWS 33	JAN 28	USGENE now provides USPTO sequence data within 3 days of publication
NEWS 34	JAN 28	TOXCENTER enhanced with reloaded MEDLINE segment
NEWS 35	JAN 28	MEDLINE and LMEDLINE reloaded with enhancements

NEWS EXPRESS 19 SEPTEMBER 2007: CURRENT WINDOWS VERSION IS V8.2,

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              CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
              AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

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=> file caplus medline embase biosis
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=> s hypertension
L1      851294 HYPERTENSION

=> s uric acid
L2       75105 URIC ACID

=> s L1 and L2
L3        6860 L1 AND L2

=> s L3 and (AY<2000 or PY<2000 or PRY<2000)
'2000' NOT A VALID FIELD CODE
'2000' NOT A VALID FIELD CODE
   2 FILES SEARCHED...
'2000' NOT A VALID FIELD CODE
'2000' NOT A VALID FIELD CODE
'2000' NOT A VALID FIELD CODE
'2000' NOT A VALID FIELD CODE
L4        3871 L3 AND (AY<2000 OR PY<2000 OR PRY<2000)

=> s xanthine oxidase
L5       38041 XANTHINE OXIDASE

=> s xanthine oxidase inhibitor
L6        3091 XANTHINE OXIDASE INHIBITOR

=> s L6 and L4
L7           9 L6 AND L4
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=> dup rem L7
PROCESSING COMPLETED FOR L7
L8 5 DUP REM L7 (4 DUPLICATES REMOVED)

=> d 1-5 L8 ibib abs

L8 ANSWER 1 OF 5 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1999335389 EMBASE
TITLE: Gout and hyperuricemia.
AUTHOR: Stanaszek M.B.
CORPORATE SOURCE: M.B. Stanaszek, c/o Dr. Walter F. Stanaszek, Health Care Consultants, 402 North Sherry Avenue, Norman, OK 73069, United States
SOURCE: Journal of Pharmacy Practice, (Aug 1999) Vol. 12, No. 4, pp. 326-334.
Refs: 25
ISSN: 0897-1900 CODEN: JPPREU
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Oct 1999
Last Updated on STN: 7 Oct 1999
AB Gout is recognized by sudden onsets of joint pain and swelling caused by imbalances in production and excretion of uric acid. Hyperuricemia is a risk factor for gout, however, not all patients with hyperuricemia will develop gout. Other risk factors include hypertension, renal insufficiency, obesity, excessive alcohol consumption, high purine diets, and medications such as thiazide diuretics and low dose aspirin. Management of gout and hyperuricemia can be achieved through inhibiting urate synthesis, enhancing urate excretion, or both. Medications to treat gout include NSAIDs, colchicine, and glucocorticosteroids. Chronic therapy with uricosuric agents or xanthine oxidase inhibitors may be necessary for those with recurrent attacks.

L8 ANSWER 2 OF 5 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1999280852 EMBASE
TITLE: Endothelial dysfunction and hypertensive vasoconstriction.
AUTHOR: De Artinano A.A.; Gonzalez V.L.-M.
CORPORATE SOURCE: A.A. De Artinano, Departamento de Farmacologia, Facultad de Medicina, Universidad Complutense de Madrid, Ciudad Universitaria s-n, 28040 Madrid, Spain
SOURCE: Pharmacological Research, (1999) Vol. 40, No. 2, pp. 113-124.
Refs: 143
ISSN: 1043-6618 CODEN: PHMREP
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 26 Aug 1999
Last Updated on STN: 26 Aug 1999
AB A change in endothelial function is a common phenomenon in patients with essential hypertension and in animals with hypertension , whether primary or induced by a salt-rich diet. In hypertensive

subjects, there may be a change in the synthesis, or the effect, of nitric oxide. Nevertheless, hypertensive vasoconstriction is at present associated, above all, with the degradation of this mediator by free radicals, such as the superoxide anion, released in the dysfunctional vascular endothelium. These radicals are also formed when hypoxanthine is turned into xanthine, and when the latter becomes uric acid, both having been catalysed by the enzyme xanthine oxidase. In physiological conditions, the concentration of superoxide radicals remains low within the organism as a result of its reaction with the superoxide dismutase enzyme. However, in pathological situations, such as arterial hypertension, there may be an increase in the production of these radicals or a deficiency of the superoxide dismutase enzyme. In hypertensive patients, the release of vasoconstrictor peroxides derived from the activity of cyclo-oxygenase in the endothelium and the vascular smooth muscle is also important. The excess free radicals released by the dysfunctional endothelium also stimulate the synthesis of these contracting agents. Moreover, it should not be forgotten that endothelin-1, which is similarly synthesized and released in the vascular endothelium, is the most powerful known endogenous vasoconstrictor. This peptide would therefore play a prominent part in some forms of hypertension. Although no changes in endothelin plasma levels have been found in essential hypertension, there may be an increase in its local concentration. It should be borne in mind that endothelin could strengthen the effect of other vasoconstrictors. Moreover, it may also provoke the release of free radicals and of cyclo-oxygenase-derived vasoconstrictor factors. The latest theories therefore indicate that the increase in vasoconstriction, which characterizes arterial hypertension, is associated with a greater production of free radicals. At the present time, antioxidant agents and xanthine oxydase-inhibiting compounds are being used to treat hypertension and other pathologies linked to endothelial dysfunction. In addition, it is thought that the therapeutic benefit of some anti-hypertensive drugs, such as calcium antagonists and angiotensin-converting enzyme inhibitors, could be in part due to the inhibition of the production of free radicals that they provoke.

L8 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 1

ACCESSION NUMBER: 1996:207673 CAPLUS
DOCUMENT NUMBER: 124:313438
TITLE: Potentiation of nitric oxide-mediated vasorelaxation by xanthine oxidase inhibitors
AUTHOR(S): Miyamoto, Yoichi; Akaike, Takaaki; Yoshida, Masaki; Goto, Shingo; Horie, Hidechika; Maeda, Hiroshi
CORPORATE SOURCE: School Medicine, Kumamoto Univ., Kumamoto, 860, Japan
SOURCE: Proceedings of the Society for Experimental Biology and Medicine (1996), 211(4), 366-73
CODEN: PSEBAA; ISSN: 0037-9727
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Nitric oxide (NO), now almost synonymous with endothelium-derived relaxing factor (EDRF), reacts with superoxide anion radical (O2-) and forms a potentially toxic mol. species, peroxynitrite (ONOO-). Because xanthine oxidase (XO) seems to be a major O2--producing enzyme in the vascular system, it is important to clarify the mechanism of XO regulation of NO/EDRF. We first characterized the inhibition of XO in vitro by three types of pyrazolopyrimidine derivs. Kinetic studies indicated that 4-amino-6-hydroxypyrazolo[3,4-d]pyrimidine (AHPP) and allopurinol competitively inhibited the conversion of xanthine to uric acid catalyzed by XO, with apparent Ki values of 0.17 ± 0.02 and 0.50 ± 0.03 μM, resp.; alloxanthine inhibited this conversion in a noncompetitive manner with an apparent Ki value of 3.54 ± 1.12 μM. O2- generation in the xanthine/XO system assayed by lucigenin-dependent chemiluminescence was suppressed most strongly by AHPP in a dose-dependent

fashion; allopurinol itself appears to reduce the enzyme by transfer of an electron to O2, thus generating O2-. AHPP significantly augmented EDRF-mediated relaxation of aortic rings from both rabbits and spontaneously hypertensive rats (SHR) in a dose-dependent manner, whereas allopurinol did not affect the relaxation and only marginal potentiation of the vasorelaxation was observed with alloxanthine. Finally, i.v. injection of AHPP (50.4 mg/kg; 100 μmol/300 g rat) reduced the blood pressure of SHR rats to 70% of the initial pressure; this pressure is almost the blood pressure of normal rats. Allopurinol (100 μmol/300 g rat; i.v.) showed transient decrease in blood pressure and moderate reduction of hypertension of SHR (10%) was observed with i.v. injection of alloxanthine (100 μmol/300 g rat). On the basis of these results, it seems that XO regulates EDRF/NO via production of O2-.

L8 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 93209170 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7681372
TITLE: Prevention and management of gout.
AUTHOR: Star V L; Hochberg M C
CORPORATE SOURCE: Department of Medicine, University of Maryland School of Medicine, Baltimore.
SOURCE: Drugs, (1993 Feb) Vol. 45, No. 2, pp. 212-22.
Ref: 35
Journal code: 7600076. ISSN: 0012-6667.
PUB. COUNTRY: New Zealand
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 14 May 1993
Last Updated on STN: 29 Jan 1996
Entered Medline: 29 Apr 1993

AB Gout is a common disease with a worldwide distribution. The major risk factor for the development of gout is sustained asymptomatic hyperuricaemia. Although pharmacological therapy of asymptomatic hyperuricaemia is not recommended, primary prevention of gout can be achieved through lifestyle changes including weight loss, restricting protein and calorie intake, limiting alcohol consumption, avoiding the use of diuretics in the treatment of hypertension, and avoiding occupational exposure to lead. The arthritis of gout can be readily managed with the use of nonsteroidal anti-inflammatory drugs (NSAIDs); systemic steroids or corticotrophin (adrenocorticotrophic hormone; ACTH) should be used in patients with contraindications to NSAIDs, or who are intolerant of them. Because of potential toxicity, colchicine should not be used to treat acute gout, but should be used in low dosage (0.6 to 1.2 mg/day) for prophylaxis of recurrent attacks of gout. The other cornerstone of prevention of recurrent gouty attacks is control of hyperuricaemia, which can be effectively accomplished with antihyperuricaemic therapy. The choice of agents, either uricosuric drugs or xanthine oxidase inhibitors, is based on the level of urinary uric acid excretion, renal function, age of patient, history of renal calculi and presence of tophi. Treatment and prevention of gout are exceedingly effective and patients can usually be managed by their primary care physician.

L8 ANSWER 5 OF 5 EMBASE
ACCESSION NUMBER: 1982065148 EMBASE
TITLE: Solid and liquid nourishment in gout. Selected historical excerpts, largely empiric or fashionable and current scientific? Concepts in the management of gout and gouty arthritis.
AUTHOR: Talbott J.H.
CORPORATE SOURCE: Arthritis Div., Dept. Med. Univ. Miami Med. Sch., Miami, FL

33101, United States
SOURCE: Seminars in Arthritis and Rheumatism, (1981) Vol. 11, No. 2, pp. 288-306.
ISSN: 0049-0172 CODEN: SAHRBF
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 019 Rehabilitation and Physical Medicine
028 Urology and Nephrology
031 Arthritis and Rheumatism
006 Internal Medicine
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB The medical literature over the centuries is abundantly supplied with extended discussions of the significance of diet and fluids, especially alcoholic beverages, in the pathogenesis and management of patients whose disease is primarily related to the content and deposition of uric acid in the body. It is recognized that uric acid is a naturally occurring substance in the body of each of us, gouty or non-gouty. A significant percentage of circulating uric acid may come from ingested sources. This amount is subject to some control. Secondary features of clinical gout, hypertension , hyperlipidemia, obesity, coronary heart disease and diabetes mellitus, may also be under partial exogenous control. During the last 25 yr, the dietary management of gout has been liberalized immeasurably by the discovery of potent uricosuric agents and a xanthine oxidase inhibitor. The result is the capability for adequate control of hyperuricemia. The persistent control of clinical arthritis may be achieved by the periodic intake of colchicine, drugs that influence uric acid metabolism, and an abundant fluid intake.

```
=> s 4 to 6 mg
L9        3685 4 TO 6 MG

=> s L4 and L9
L10          0 L4 AND L9

=> s 6 mg/dL
'DL' IS NOT A VALID FIELD CODE
'DL' IS NOT A VALID FIELD CODE
'DL' IS NOT A VALID FIELD CODE
'DL' IS NOT A VALID FIELD CODE
L11          0 6 MG/DL

=> s 6 mg
L12      91746 6 MG

=> s L12 and L4
L13         50 L12 AND L4

=> dup rem L13
PROCESSING COMPLETED FOR L13
L14          27 DUP REM L13 (23 DUPLICATES REMOVED)

=> d 20-27 L14 ibib abs
```

L14 ANSWER 20 OF 27 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1985077477 EMBASE
TITLE: Multicentre randomized cross-over double-blind comparison between chlorthalidone and slow-release oxprenolol in mild-to-moderate hypertension.
AUTHOR: Pollavini G.; Comi D.; Grillo C.; et. al.

CORPORATE SOURCE: Interdisciplinary Group for the Prevention and Treatment of Arterial Hypertension, 20133 Milan, Italy
SOURCE: Current Therapeutic Research - Clinical and Experimental, (1984) Vol. 35, No. 3, pp. 467-475.
ISSN: 0011-393X CODEN: CTCEA9
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB In a formal multicentre study, 298 hypertensive out-patients were given both chlorthalidone 25 mg once daily (o.d.) and slow-release oxprenolol 160 mg 0.d. for 4 weeks each, according to a randomized double-blind crossover protocol. Each active treatment period was preceded by a 1-month single-blind placebo treatment. Both chlorthalidone and slow-release oxprenolol elicited a good antihypertensive effect, but the heart rate decreased only on slow-release oxprenolol. Supine diastolic blood pressure at rest fell below 95 mmHg in 50% of patients after slow-release oxprenolol, and in 55% of patients after chlorthalidone. There was no statistical or clinical difference between the two drugs in terms of antihypertensive activity. Whereas none of the laboratory parameters varied significantly after slow-release oxprenolol, chlorthalidone treatment was associated with a 3 mg/100 ml increase in serum glucose, a 0.7 mg/100 ml increase in serum uric acid, a 6 mg/100 ml increase in serum cholesterol, and a fall in serum potassium of 0.3 mEq/l. All these metabolic changes were statistically significant, but considering both single and mean values, no conclusion in terms of possible clinical relevance, can be drawn. Unwanted effects, both on slow-release oxprenolol and on chlorthalidone, were few and never required withdrawal from the study. Tolerability was similar on both treatments. In conclusion, slow-release oxprenolol 160 mg daily and chlorthalidone 25 mg daily appear to be equivalent in terms of antihypertensive potency, but the diuretic may induce some biochemical changes which, although not of considerable clinical relevance, may require further studies to be better understood.

L14 ANSWER 21 OF 27 MEDLINE on STN DUPLICATE 13
ACCESSION NUMBER: 84209386 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6373442
TITLE: A double-blind multicentre study of piretanide and hydrochlorothiazide in patients with essential hypertension.
AUTHOR: Buckert C; Muhlhausler W; Fratzer U; Verho M
SOURCE: The Journal of international medical research, (1984) Vol. 12, No. 2, pp. 81-6.
Journal code: 0346411. ISSN: 0300-0605.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
(COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198407
ENTRY DATE: Entered STN: 20 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 2 Jul 1984

AB In a randomized double-blind parallel group study conducted in three centres the hypotensive activity of piretanide 6 mg b.i.d. was compared with that of hydrochlorothiazide (HCT) 25 mg b.i.d.

and HCT 50 mg b.i.d. Ninety-three patients entered the study and sixty-one completed a 16-week trial period. All three treatments produced a significant reduction in supine diastolic and systolic blood pressure after only 2 weeks of active treatment and this was maintained for the duration of the study. The mean maximal reduction in supine diastolic blood pressure was 18% in the piretanide group, 18.8% in the HCT 25 mg b.i.d. group, and 20% in the HCT 50 mg b.i.d. group. The corresponding figures for the percentage of patients attaining a supine diastolic pressure below 95 mm Hg were 83%, 62% and 89%. There were no significant differences between the three groups. Side-effects were generally mild and transient, except for polyuria which was noted in all three groups but more commonly in the piretanide group. Two patients were withdrawn because of side-effects: one patient in the high dose HCT group developed severe postural symptoms; and one patient in the low dose HCT group was withdrawn because of restlessness, nausea, weakness, dizziness and somnolence. All three treatments caused a significant increase in serum uric acid concentrations. Four patients in each of the HCT groups developed hypokalaemia, but no patients in the piretanide group did so.

L14 ANSWER 22 OF 27 MEDLINE on STN
ACCESSION NUMBER: 82262707 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6921124
TITLE: [The value of a single determination of serum uric acid concentration in the early diagnosis of hypertensive disorders in pregnancy (author's transl)]. Die Aussagekraft einer einmaligen Bestimmung der Harnsaurekonzentration im Serum zur Fruherfassung der hypertensiven Schwangerschaftskomplikationen.
AUTHOR: Oney T; Kaulhausen H; Schlebusch H
SOURCE: Geburtshilfe und Frauenheilkunde, (1982 Jun) Vol. 42, No. 6, pp. 440-3.
Journal code: 0370732. ISSN: 0016-5751.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198210
ENTRY DATE: Entered STN: 17 Mar 1990
Last Updated on STN: 17 Mar 1990
Entered Medline: 29 Oct 1982

AB In 200 healthy, normotensive nulliparous women, a single determination of serum uric acid concentration was done between weeks 28-32 of gestation in order to identify a possible increased risk of developing hypertensive complications. If serum concentrations higher than 3.6 mg/dl were considered as increased ("positive test"), women who developed toxaemia of late pregnancy (proteinuric hypertension), had a significantly elevated mean serum uric acid concentration already at the beginning of the third trimester (p less than 0.01). The incidence of toxaemia and hypertensive disease without proteinuria was significantly higher in the group of women with an elevated uric acid value (p less than 0.001). Only 9% of pregnant women with a "negative test" ultimately developed a mild form of a hypertensive complication. Conversely, 74% of the patients with a "positive test" remained normotensive. Thus, the predictive value of a "positive test" is low (26%) and that of a "negative test" relatively high (91%).

L14 ANSWER 23 OF 27 EMBASE DUPLICATE 14
ACCESSION NUMBER: 1982107594 EMBASE
TITLE: Biochemical cardiac risk factors during diuretic therapy.
AUTHOR: Hubbell F.A.; Weber M.A.; Winer R.L.; Rose D.E.

CORPORATE SOURCE: Sect. Clin. Pharmacol. Hypertens., Veterans Adm. Med. Cent., Long Beach, CA, United States
SOURCE: Archives Internationales de Pharmacodynamie et de Therapie, (1982) Vol. 256, No. 1, pp. 123-133.
ISSN: 0003-9780 CODEN: AIPTAK
COUNTRY: Belgium
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

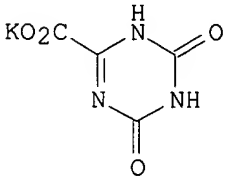
AB The effects of six weeks of treatment with fixed doses of the diuretics chlorthalidone and tricrynafen on serum total cholesterol, low density lopoproteins, high density lipoproteins, triglycerides and other biochemical parameters were studied in 24 hypertensive patients. Chlorthalidone increased total serum cholesterol by 40 ± 11 (SEM) mg/dl (P<0.01) and triglycerides by 80 ± 30 mg/dl (P<0.05). The increase of 16 ± 21 mg/dl in low density lipoproteins and the decrease of 5 ± 6 mg/dl in high density lipoproteins induced by this drug were not singificant. An investigational uricosuric diuretic, ticrynafen, caused no significant changes in any of the serum lipid concentrations measured. Fasting blood glucose levels were elevated, but not significantly, by both diuretics. Serum uric acid concentrations tended to increase in patients given chlorthalidone while ticrynafen treatment decreased uric acid levels by 2.9 ± 1.4 mg/dl (P<0.01). The elevations induced by chlorthalidone in serum lipid levels may partially counteract the potential benefits of its antihypertensive effects.

L14 ANSWER 24 OF 27 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 1981:214900 BIOSIS
DOCUMENT NUMBER: PREV198171084892; BA71:84892
TITLE: DIABETES MELLITUS HYPERTENSION AND BETA BLOCKING AGENTS PINDOLOL AND OXPRENOLOL.
AUTHOR(S): JOVANOVIC D [Reprint author]
CORPORATE SOURCE: REGIONAL MED CENT 23, AVGUST DOBOJ, INT MED SERVICE, DIABETIC CLINIC
SOURCE: Diabetologia Croatica, (1980) Vol. 9, No. 3, pp. 313-322.
CODEN: DBCRB2. ISSN: 0351-0042.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: SERBO-CROATIAN; ENGLISH

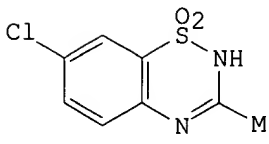
AB Observation on 120 diabetic patients with grade I, II and borderline II/III hypertension on pindole therapy [once daily, mean dose 12.6 ± 2.0 mg/day] and on oxprenolol [mean dose 60 ± 19.6 mg/day], a favorable decrease in arterial pressure was observed: -22 ± 14 mm Hg for systolic and -17 ± 3 mm Hg for diastolic, accompanied by a decrease of -2.43 ± 2.39 mmol/l (mean ± SD) in glucose levels. The decreased glucose levels were more significant in the first 6 weeks. Cholesterol and triglycerides had a tendency toward normal values, but without the significant changes for total lipids and uric acid. One daily dose of pindolol and oxprenolol does not present a particular risk for diabetics with hypertension , although more frequent controls in stable, mild and moderate diabetes mellitus are necessary.

L14 ANSWER 25 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 15
ACCESSION NUMBER: 1979:551263 CAPLUS
DOCUMENT NUMBER: 91:151263
ORIGINAL REFERENCE NO.: 91:24269a,24272a

TITLE: The oxonate pretreated rat as a model for evaluating hyperuricemic effects of antihypertensive drugs
AUTHOR(S): Smith, R. D.; Essenburg, A. D.; Kaplan, H. R.
CORPORATE SOURCE: Pharm. Res. Div., Warner-Lambert/Parke-Davis, Ann Arbor, MI, 48105, USA
SOURCE: Clinical and Experimental Hypertension (1978-1981) (1979), 1(4), 487-504
CODEN: CEHYDQ; ISSN: 0148-3927
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I



II

AB The K oxonate (I) [2207-75-2]-pretreated (uricase-inhibited) rat was evaluated as an in vivo model in which to test the hyperuricemic effect of antihypertensive drugs. Inhibition of uricase by I pretreatment (2 + 250 mg/kg i.p.) elevated plasma uric acid [69-93-2] levels. Allopurinol (50 mg/kg orally) blocked the I-induced elevation of endogenously synthesized uric acid but had no effect on the response to exogenously administered uric acid. In the I-pretreated rat diazoxide (II) [364-98-7] (50 mg/kg i.p.) produced a significant elevation in plasma uric acid levels which was blocked by allopurinol. Since II reduced the urinary uric acid excretion, a renal effect is also implicated. Other agents used in the treatment of hypertension such as hydralazine [86-54-4] (6 mg/kg), furosemide [54-31-9] (100 mg/kg), and prazosin [19216-56-9] (10 mg/kg) produced small but significant increases in plasma uric acid levels in rats pretreated with I when administered i.p. The doses of these agents required to produce the hyperuricemic response were greater than those required to produce their characteristics diuretic or antihypertensive effects. The hyperuricemic effects of all 3 agents were blocked by allopurinol. Thus, the I-pretreated rat can be used to study the effects on uric acid synthesis in vivo. The data with hydralazine and prazosin, however, showing a hyperuricemia in I-pretreated rats when none has been observed during the clin. use of these compds., raises serious question as to its utility as a model for predicting the hyperuricemic liability of new drugs.

L14 ANSWER 26 OF 27 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 1980:211852 BIOSIS
DOCUMENT NUMBER: PREV198070004348; BA70:4348
TITLE: EFFECTS OF ANTI HYPERTENSIVE DRUGS ON DIALYSIS RESISTANT HYPERTENSION PLASMA RENIN AND DOPAMINE BETA HYDROXYLASE ACTIVITIES METABOLIC RISK FACTORS AND CALCIUM PHOSPHATE HOMEOSTASIS COMPARISON OF METOPROLOL ALPHA METHYL DOPA AND CLONIDINE IN A CROSSOVER TRIAL.
AUTHOR(S): DE FREMONT J F [Reprint author]; COEVOET B; ANDREJAK M; MAKDASSI R; QUICHAUD J; LAMBREY G; GUERIS J; CAILLENS C; HARICHAUX P; ET AL
CORPORATE SOURCE: FAC MED HOP, CENT HOSP REG AMIENS, AMIENS, FR

SOURCE: Clinical Nephrology, (1979) Vol. 12, No. 5, pp. 198-205.
CODEN: CLNHBI. ISSN: 0301-0430.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB To establish the choice of antihypertensive drugs in the treatment of dialysis resistant hypertension, the clinical efficiency and side effects, together with the effects on metabolite vascular risk factors (blood levels of glucose (G), cholesterol (CH), triglycerides (TG), uric acid (UA), Ca and phosphate (PO4)) were assessed in 13 patients with hypertension resistant to volume depletion. For periods of 6 wk these patients received, in randomized sequences, metoprolol (M), α methyldopa (A) and clonidine (C) in doses sufficient to achieve normal blood pressure (BP) wihout any other therapeutic change, and without any change in dry weight. Assessment, made at the 1st dialysis of the 6th wk, using covariance analysis, showed no significant difference between the 3 drugs as regards pre- and post-dialysis supine BP, plasma renin (PRA) and dopamine betahydroxylase (DBH) activities, predialysis standing BP and post-dialysis heart rate (HR). Post-dialysis standing mean BP was lower with A than with M or C. Predialysis HR was lower with M than with A and C. There was no difference between the 3 treatments in the incidence of hypertension during dialysis, but the incidence of hypotension was greater with A (13% of the dialyses) than with M (5%) or C (6.5%). Constipation, mouth dryness and drowsiness were more frequent with clonidine. Acute pulmonary edema occurred in 3 cases with cardiac enlargement while they were on metoprolol and led to discontinuation of this drug. Predialysis G was higher with C (111 ± 5) than with M (98 ± 4) or A (92 ± 6 mg/dl). Predialysis CH was lower with M (160 ± 8 mg/dl) than with A or C (185 ± 8). Predialysis Ca was higher with C (9.2 ± 2 mg/dl) than with M (8.5 ± 0.2). No difference was found for TG, K, UA, plasma osmolality, parathyroid hormone or calcitonin. There are no clinically imperative reasons to prefer 1 drug to the other except for the existence of cardiomegaly which is a contraindication to metoprolol and an increased susceptibility to hypotension which is a contraindication to αmethyldopa. The differences seen in the metabolic risk factors (higher blood G and P [plasma] Ca with clonidine, and lower blood CH with metoprolol) would favor the use of metoprolol, but the long-term prognostic significance of the differences are not established. Inhibition of renin secretion should not be considered as a reason to prefer β blockers like metoprolol; at equally antihypertensive doses, metoprolol did not suppress renin secretion more efficiently than α methyldopa or clonidine. Post-dialysis orthostatic hypotension seen only with α methyldopa and the higher incidence of hypotension during dialysis seen with this drug are not explained by different plasma DBH and renin activities.

L14 ANSWER 27 OF 27 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1965:419724 CAPLUS
DOCUMENT NUMBER: 63:19724
ORIGINAL REFERENCE NO.: 63:3523e-g
TITLE: Intensified hypertension therapy with a combination treatment of Lasix (fursemide) and reserpine
AUTHOR(S): Engelmann, G.
SOURCE: Muenchener Medizinische Wochenschrift (1965), 107(21), 1060-3
CODEN: MMWOAU; ISSN: 0027-2973
DOCUMENT TYPE: Journal
LANGUAGE: German

AB The synergistic action of Lasix-reserpine combination treatment has been demonstrated in 100 patients with hypertension of various etiology and with varying degree of severity. The hypotensive action of

this combination considerably exceeded the well known effects of the two single substances. A daily dosage of 20-60 mg. of Lasix and 0.2-0. 6 mg. of reserpine caused a systolic decrease of 14.4% and a diastolic decrease of 15.2%. This combination was particularly effective in very high mean initial values. There was no effect of the medication on the K, Na, creatinine, and uric acid levels.

```
=> s high blood pressure
L15      21560 HIGH BLOOD PRESSURE

=> s uric acid
L16      75105 URIC ACID

=> s L15 and L16
L17        262 L15 AND L16

=> s L17 and (AY<2000 or PY<2000 or PRY<2000)
'2000' NOT A VALID FIELD CODE
'2000' NOT A VALID FIELD CODE
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'2000' NOT A VALID FIELD CODE
'2000' NOT A VALID FIELD CODE
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L18        158 L17 AND (AY<2000 OR PY<2000 OR PRY<2000)

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PROCESSING COMPLETED FOR L18
L19         106 DUP REM L18 (52 DUPLICATES REMOVED)

=> s allopurinol
L20      26781 ALLOPURINOL

=> s L19 and L20
L21          0 L19 AND L20

=> s xanthine oxidase inhibitor
L22       3091 XANTHINE OXIDASE INHIBITOR

=> s L19 and L22
L23          0 L19 AND L22

=> s L17 and L22
L24          0 L17 AND L22

=> s L17 and L20
L25          5 L17 AND L20

=> dup rem L25
PROCESSING COMPLETED FOR L25
L26           3 DUP REM L25 (2 DUPLICATES REMOVED)

=> d 1-3 L26 ibib abs

L26  ANSWER 1 OF 3  EMBASE  COPYRIGHT (c) 2008 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:   2007211003  EMBASE
TITLE:              Evaluation of hypertension by the general pediatrician.
AUTHOR:             Nguyen M.; Mitsnefes M.
CORPORATE SOURCE:   Dr. M. Mitsnefes, Nephrology and Hypertension, Cincinnati
                    Children's Hospital Medical Center, MLC 7022, 3333 Burnet
                    Ave, Cincinnati, OH 45229, United States.
                    mark.mitsnefes@cchmc.org
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SOURCE: Current Opinion in Pediatrics, (Apr 2007) Vol. 19, No. 2, pp. 165-169.
Refs: 27
ISSN: 1040-8703 CODEN: COPEE9
PUBLISHER IDENT.: 00008480200704000000009
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
007 Pediatrics and Pediatric Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 21 May 2007
Last Updated on STN: 21 May 2007

AB PURPOSE OF REVIEW: To summarize the recommended work-up in a child who presents with elevated blood pressure as well as innovative evaluation techniques under development. RECENT FINDINGS: 'The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents' contains several updates on the diagnosis, evaluation and treatment of childhood hypertension. New risk factors for hypertension have been identified and include obesity, sleep apnea, and low birth weight. The roles of uric acid, leptin and C-reactive protein in the pathophysiology of hypertension have been examined. The presence of hypertensive end-organ damage has been demonstrated in hypertensive children. SUMMARY: Current knowledge emphasizes the need to diagnose and treat hypertension when it develops in childhood to decrease the risk of cardiovascular morbidity in adulthood. End-organ injury is evident, illustrated by the presence of left ventricular hypertrophy, even in young children. Assessment for the presence of comorbidities and end-organ damage should be emphasized. Further study is needed to isolate the etiologic factors for childhood hypertension, improve evaluation techniques, and determine if end-organ damage is reversible with proper therapy. .COPYRGT. 2007 Lippincott Williams & Wilkins, Inc.

L26 ANSWER 2 OF 3 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2007245530 EMBASE
TITLE: Novel concepts in the pathogenesis and management of pediatric hypertension.
AUTHOR: Silverstein D.M.
CORPORATE SOURCE: D.M. Silverstein, Children's Hospital New Orleans, Division of Nephrology, 200 Henry Clay Avenue, New Orleans, LA 70115, United States. dsilve@lsuhsc.edu
SOURCE: Current Pediatric Reviews, (May 2007) Vol. 3, No. 2, pp. 109-114.
Refs: 94
ISSN: 1573-3963
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
005 General Pathology and Pathological Anatomy
007 Pediatrics and Pediatric Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Jul 2007
Last Updated on STN: 9 Jul 2007

AB Hypertension is a major cause of morbidity and mortality throughout the world and an independent risk factor for cardiovascular disease. Although the incidence of hypertension is significantly greater in adults than children, recent trends reveal a rising percentage of children with high blood pressure. Coincident with the dramatic increase of the number of children with hypertension has been a

growing field of knowledge regarding novel causes and therapeutic options for children with high blood pressure. Most reviews in pediatric hypertension focus on the many traditional causes of hypertension in children including renovascular disease, renal parenchymal disease, medication usage, endocrine causes (e.g. hyperthyroidism), and cardiovascular causes (e.g. coarctation of the aorta). However, recent research suggests that non-traditional causes of hypertension including chronic inflammation, low nephron number, prematurity/low birth weight, malnutrition, obesity (as part of the metabolic syndrome), hyperinsulinemia/insulin resistance, elevated uric acid , and dietary factors may be more common than previously thought. There are also innovative concepts in the treatment of childhood hypertension, including behavioral and combination drug therapy. In a rapidly evolving field and epidemic of children developing high blood pressure, it is therefore imperative for the generalist and specialist to be cognizant of the many changes occurring in the pathogenesis and management of childhood hypertension. .COPYRGT. 2007 Bentham Science Publishers Ltd.

L26 ANSWER 3 OF 3 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2005530043 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16206348
TITLE: Factors associated with musculoskeletal disability and chronic renal failure in clinically diagnosed primary gout.
AUTHOR: Alvarez-Nemegyei Jose; Cen-Piste Julio Cesar; Medina-Escobedo Martha; Villanueva-Jorge Salha
CORPORATE SOURCE: Instituto Mexicano del Seguro Social, Calle 57 # 503 x 50 y 62, Col. Centro, 97000 Merida, Yucatan, Mexico.. nemegyei@hotmail.com
SOURCE: The Journal of rheumatology, (2005 Oct) Vol. 32, No. 10, pp. 1923-7.
Journal code: 7501984. ISSN: 0315-162X.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 6 Oct 2005
Last Updated on STN: 18 Dec 2005
Entered Medline: 13 Dec 2005

AB OBJECTIVE:. To assess the association between a set of sociodemographic, clinical, and biochemical variables and the presence of musculoskeletal (MSK) disability and chronic renal failure in patients with primary gout defined using Wallace criteria. METHODS: Subjects were 90 patients with primary gout (98% male, age 54 +/- 12 years, 11.3 +/- 9.8 years with gout). A cohort nested case-control design was used. Analysis was done of the association between MSK disability or renal failure and a series of variables: age; duration of gout; body mass index; education level; income; serum glucose, cholesterol, triglycerides, and uric acid; Health Assessment Questionnaire score; obesity; family history of gout; high blood pressure; alcoholism; smoking habit; presence of tophi; ischemic cardiopathy; and use of colchicine, glucocorticoids, nonsteroidal antiinflammatory drug, or allopurinol. RESULTS: Forty-two patients (47%) had MSK disability, and 25/80 (31%) had renal failure. On logistic regression, presence of tophi (relative risk 4.3, 95% confidence interval 1.2-15.1), hypertriglyceridemia (RR 3.4, 95% CI 1.1-10), and history of ischemic heart disease (RR 8.3, 95% CI 1.6-41) were associated with MSK disability. Patient age was the only variable associated with renal failure. CONCLUSION: Optimal medical control of gout and its comorbidities may improve prognosis of gout, as suggested by our findings, in which a marker for poorly controlled gout such as presence of tophi in addition to high blood triglyceride levels and ischemic heart disease were associated with MSK disability. Older age was the only factor associated with renal failure, although this may only reflect declining renal function in the

elderly.

=> s hypertension
L27 851294 HYPERTENSION

=> s L16 and L27
L28 6860 L16 AND L27

=> s allopurinol
L29 26781 ALLOPURINOL

=> s L28 and L29
L30 353 L28 AND L29

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L31 122 L30 AND (AY<2000 OR PY<2000 OR PRY<2000)

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PROCESSING COMPLETED FOR L31
L32 102 DUP REM L31 (20 DUPLICATES REMOVED)

=> d 1-10 L32 ibib abs

L32 ANSWER 1 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1999335389 EMBASE
TITLE: Gout and hyperuricemia.
AUTHOR: Stanaszek M.B.
CORPORATE SOURCE: M.B. Stanaszek, c/o Dr. Walter F. Stanaszek, Health Care Consultants, 402 North Sherry Avenue, Norman, OK 73069, United States
SOURCE: Journal of Pharmacy Practice, (Aug 1999) Vol. 12, No. 4, pp. 326-334.
Refs: 25
ISSN: 0897-1900 CODEN: JPPREU
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Oct 1999
Last Updated on STN: 7 Oct 1999
AB Gout is recognized by sudden onsets of joint pain and swelling caused by imbalances in production and excretion of uric acid. Hyperuricemia is a risk factor for gout, however, not all patients with hyperuricemia will develop gout. Other risk factors include hypertension, renal insufficiency, obesity, excessive alcohol consumption, high purine diets, and medications such as thiazide diuretics and low dose aspirin. Management of gout and hyperuricemia can be achieved through inhibiting urate synthesis, enhancing urate excretion, or both. Medications to treat gout include NSAIDs, colchicine, and glucocorticosteroids. Chronic therapy with uricosuric agents or xanthine oxidase inhibitors may be necessary for those with recurrent attacks.

L32 ANSWER 2 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights

reserved on STN
ACCESSION NUMBER: 1998117157 EMBASE
TITLE: 'Bad dietary habits' and recurrent calcium oxalate nephrolithiasis.
AUTHOR: Hess B.
CORPORATE SOURCE: Dr. B. Hess, Department of Medicine, University Hospital, CH-3010 Berne, Switzerland
SOURCE: Nephrology Dialysis Transplantation, (Apr 1998) Vol. 13, No. 4, pp. 1033-1038.
Refs: 33
ISSN: 0931-0509 CODEN: NDTREA
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 028 Urology and Nephrology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
006 Internal Medicine
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Apr 1998
Last Updated on STN: 29 Apr 1998

L32 ANSWER 3 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1998339580 EMBASE
TITLE: Gout in the elderly. Clinical presentation and treatment.
AUTHOR: Fam A.G.
CORPORATE SOURCE: Dr. A.G. Fam, Sunnybrook Health Science Centre, 2075 Bayview Avenue, Toronto, Ont. M4N 3M5, Canada
SOURCE: Drugs and Aging, (1998) Vol. 13, No. 3, pp. 229-243.
Refs: 74
ISSN: 1170-229X CODEN: DRAGE6
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 020 Gerontology and Geriatrics
030 Clinical and Experimental Pharmacology
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 22 Oct 1998
Last Updated on STN: 22 Oct 1998

AB Gout in the elderly differs from classical gout found in middle-aged men in several respects: it has a more equal gender distribution, frequent polyarticular presentation with involvement of the joints of the upper extremities, fewer acute gouty episodes, a more indolent chronic clinical course, and an increased incidence of tophi. Long term diuretic use in patients with hypertension or congestive cardiac failure, renal insufficiency, prophylactic low dose aspirin (acetylsalicylic acid), and alcohol (ethanol) abuse (particularly by men) are factors associated with the development of hyperuricaemia and gout in the elderly. Extreme caution is necessary when prescribing nonsteroidal anti-inflammatory drugs (NSAIDs) for the treatment of acute gouty arthritis in the elderly. NSAIDs with short plasma half-life (such as diclofenac and ketoprofen) are preferred, but these drugs are not recommended in patients with peptic ulcer disease, renal failure, uncontrolled hypertension or cardiac failure. Colchicine is poorly tolerated in the elderly and is best avoided. Intra-articular and systemic corticosteroids are increasingly being used for treating acute gouty flares in aged patients with medical disorders contraindicating NSAID therapy. Urate-lowering drugs are indicated for the treatment of hyperuricaemia and chronic gouty arthritis. Uricosuric drugs are poorly tolerated and the frequent presence of renal impairment in the elderly renders these drugs ineffective. Allopurinol is the urate-lowering drug of choice, but its use in the aged is associated with an increased incidence of both

cutaneous and severe hypersensitivity reactions. To minimise this risk, allopurinol dose must be kept low. A starting dose of allopurinal 50 to 100 mg on alternate days, to a maximum daily dose of about 100 to 300 mg, based upon the patient's creatinine clearance and serum urate level, is recommended. Asymptomatic hyperuricaemia is not an indication for long term urate-lowering therapy; the risks of drug toxicity often outweigh any benefit.

L32 ANSWER 4 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1998293223 EMBASE
TITLE: Gout accompanying rheumatoid arthritis: A comparison of affected women and men.
AUTHOR: Wooten M.D.; Lipsmeyer E.
CORPORATE SOURCE: Dr. M.D. Wooten, Section of Rheumatology, Veterans Admin. Medical Center, 1601 Kirkwood Highway, Wilmington, DE 19805, United States
SOURCE: Journal of Clinical Rheumatology, (Aug 1998) Vol. 4, No. 4, pp. 220-224.
Refs: 38
ISSN: 1076-1608 CODEN: JCRHFM
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Sep 1998
Last Updated on STN: 17 Sep 1998

AB The coexistence of rheumatoid arthritis and gout has been recognized as a rare event. We report a case of a premenopausal woman who was discovered to have tophaceous gout at age 41 after having had rheumatoid arthritis for 16 years. She developed nephrotic syndrome from auranofin and renal insufficiency from hypertension, which led to hyperuricemia. Twenty-two cases of coexistent rheumatoid arthritis and gout have been reported, and 16 were in men, whereas six were in women. Women were more likely than men to develop rheumatoid arthritis first, whereas men were more likely to develop gout first. Both tophi and rheumatoid nodules are common in these patients, with tophi being slightly more common in women and more often found on the hands. No statistically significant differences exist in the serum uric acid levels of women with both types of arthritis compared with men. Renal insufficiency is a risk factor for the subsequent development of gout in women with rheumatoid arthritis. Both rheumatoid arthritis and gout should be investigated for in patients with features suggestive of both diseases.

L32 ANSWER 5 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1997177664 EMBASE
TITLE: Intradermal urate tophi.
AUTHOR: Fam A.G.; Assaad D.
CORPORATE SOURCE: Dr. A.G. Fam, Division of Rheumatology, Sunnybrook Health Science Centre, University of Toronto, 2075 Bayview Avenue, Toronto, Ont. M4N 3M5, Canada
SOURCE: Journal of Rheumatology, (Jun 1997) Vol. 24, No. 6, pp. 1126-1131.
Refs: 15
ISSN: 0315-162X CODEN: JRHUA9
COUNTRY: Canada
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
031 Arthritis and Rheumatism
037 Drug Literature Index
006 Internal Medicine

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jul 1997
Last Updated on STN: 23 Jul 1997

AB Objective. To analyze the clinical features and identify risk factors associated with the development of intradermal urate tophi. Methods. Six patients (5 men and 1 woman, mean age 59.8 yrs) with intradermal tophi were studied between 1987 and 1996. Results. Intradermal urate crystal deposits appeared as small, superficial, pustule-like, whitish lesions. All patients experienced superimposed inflammatory episodes with increasing pain, swelling, and erythema of the intradermal tophi. In one patient, the lesions were associated with a peculiar skin hyperpigmentation. Five had intermittent liquefaction and ulcerations of the lesions with drainage of white chalky matter from which monosodium urate crystals were recovered. Mean pre-treatment serum urate was 570.6 μmol/l (range 496-720). Risk factors for gout and intradermal tophi included renal failure in all 6, hypertension and chronic diuretic therapy in 4, and one patient each with alcohol abuse, chronic low dose acetylsalicylic acid, myeloma, and a positive family history. Conclusion. Intradermal urate tophi with superimposed inflammatory episodes, intermittent ulcerations, and possibly pigmentary changes, are rare skin manifestations of chronic tophaceous gout. Renal insufficiency, hypertension, and chronic diuretic use are factors associated with the development of hyperuricemia and gout in these patients.

L32 ANSWER 6 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1997304267 EMBASE
TITLE: Thiazide diuretics and the initiation of anti-gout therapy.
AUTHOR: Gurwitz J.H.; Kalish S.C.; Bohn R.L.; Glynn R.J.; Monane M.; Mogun H.; Avorn J.
CORPORATE SOURCE: Dr. J.H. Gurwitz, Meyers Primary Care Institute, 100 Central Street, Worcester, MA 01608, United States
SOURCE: Journal of Clinical Epidemiology, (Aug 1997) Vol. 50, No. 8, pp. 953-959.
Refs: 36
ISSN: 0895-4356 CODEN: JCEPEE
PUBLISHER IDENT.: S 0895-4356(97)00101-7
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Oct 1997
Last Updated on STN: 23 Oct 1997

AB While physiologic and epidemiologic evidence link diuretic therapy with hyperuricemia, no previous study has quantified the risk for initiation of treatment specific for hyperuricemia or gout among elderly patients taking thiazide diuretics. We performed a retrospective cohort study of 9249 enrollees aged 65 or older in the New Jersey Medicaid program who were newly started on an antihypertensive medication from November 1981 through February 1989 and who had no prier use of anti gout therapy (allopurinol, colchicine, or a uricosuric) during the preceding one-year period. We used Cox proportional hazards analysis to determine the risk for the initiation of anti gout therapy in patients using various antihypertensive treatment regimens relative to no antihypertensive exposure. Patient follow-up extended for up to two years. Antihypertensive exposure was characterized over the entire period of follow-up according to the following categories: thiazide diuretic therapy alone; non-thiazide antihypertensive therapy; thiazide diuretic therapy in combination with any non-thiazide antihypertensive agent(s); and no antihypertensive use. Antihypertensive exposure was entered into the

model as a time-varying covariate. Estimates of risk were adjusted fur age, Sex, race, nursing home residence, number of prescriptions filled, intensity of physician use, hospitalization history, and year of antihypertensive treatment initiation. The adjusted relative risk for the initiation of anti-gout therapy was 1.00 (95% CI, 0.65-1.53) for non-thiazide antihypertensive therapy alone, 1.99 (95% CI, 1.21-3.26) for thiazide diuretic therapy, and 2.29 (95% CI, 1.55-3.37) for thiazide diuretic therapy in combination with any non-thiazide agent(s). Risk for antigout therapy was significantly increased for thiazide doses of ≤ 25 mg/day (in hydrochlorothiazide equivalents); no significant increase in risk was seen for lower doses. We conclude that use of thiazide diuretics in doses of 25 mg/day or higher is associated with a significantly increased risk for initiation of anti-gout therapy. Such treatment may reflect the occurrence of clinical sequelae of diuretic-induced hyperuricemia or the inappropriate treatment of asymptomatic hyperuricemia.

L32 ANSWER 7 OF 102 MEDLINE on STN
ACCESSION NUMBER: 97253410 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9098853
TITLE: Purine metabolism and inhibition of xanthine oxidase in severely hypoxic neonates going onto extracorporeal membrane oxygenation.
AUTHOR: Marro P J; Baumgart S; Delivoria-Papadopoulos M; Zirin S; Corcoran L; McGaurn S P; Davis L E; Clancy R R
CORPORATE SOURCE: Children's Hospital of Philadelphia, Pennsylvania, USA.
CONTRACT NUMBER: HD-20337 (United States NICHD)
N01-NS-1-2315 (United States NINDS)
RR-00240 (United States NCRR)
SOURCE: Pediatric research, (1997 Apr) Vol. 41, No. 4 Pt 1, pp. 513-20.
Journal code: 0100714. ISSN: 0031-3998.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 16 Jul 1997
Last Updated on STN: 16 Jul 1997
Entered Medline: 2 Jul 1997

AB The effect of allopurinol to inhibit purine metabolism via the xanthine oxidase pathway in neonates with severe, progressive hypoxemia during rescue and reperfusion with extracorporeal membrane oxygenation (ECMO) was examined. Twenty-five term infants meeting ECMO criteria were randomized in a double-blinded, placebo-controlled trial. Fourteen did not receive allopurinol, whereas 11 were treated with 10 mg/kg after meeting criteria and before cannulation, in addition to a 20-mg/kg priming dose to the ECMO circuit. Infant plasma samples before cannulation, and at 15, 30, 60, and 90 min, and 3, 6, 9, and 12 h on bypass were analyzed (HPLC) for allopurinol, oxypurinol, hypoxanthine, xanthine, and uric acid concentrations. Urine samples were similarly evaluated for purine excretion. Hypoxanthine concentrations in isolated blood-primed ECMO circuits were separately measured. Hypoxanthine, xanthine, and uric acid levels were similar in both groups before ECMO. Hypoxanthine was higher in allopurinol-treated infants during the time of bypass studied ($p = 0.022$). Xanthine was also elevated ($p < 0.001$), and uric acid was decreased ($p = 0.005$) in infants receiving allopurinol. Similarly, urinary elimination of xanthine increased ($p < 0.001$), and of uric acid decreased ($p = 0.04$) in treated infants. No allopurinol toxicity was observed. Hypoxanthine concentrations were significantly higher in isolated ECMO

circuits and increased over time during bypass ($p < 0.001$). This study demonstrates that allopurinol given before cannulation for and during ECMO significantly inhibits purine degradation and uric acid production, and may reduce the production of oxygen free radicals during reoxygenation and reperfusion of hypoxic neonates recovered on bypass.

L32 ANSWER 8 OF 102 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 1997:337113 CAPLUS
DOCUMENT NUMBER: 127:16211
TITLE: Acceleration of hypertensive cerebral injury by the inhibition of xanthine-xanthine oxidase system in stroke-prone spontaneously hypertensive rats
AUTHOR(S): Maenishi, Osamu; Ito, Hiroyuki; Suzuki, Tsuneyuki
CORPORATE SOURCE: Department of Pathology, Kinki University School of Medicine, Osaka, 589, Japan
SOURCE: Clinical and Experimental Hypertension (1997), 19(4), 461-477
CODEN: CEHYER; ISSN: 1064-1963
PUBLISHER: Dekker
DOCUMENT TYPE: Journal
LANGUAGE: English
AB It is well-known that, in ischemic cerebral injury, a free radical and its byproducts are generated by xanthine-xanthine oxidase system and eliminated by scavengers such as superoxide dismutase (SOD), catalase, uric acid and ascorbic acid. To investigate the possible involvement of the xanthine-xanthine oxidase system in hypertensive cerebral injury, the authors examined chronol. changes in uric acid level in the cerebral cortex and the effects of the inhibition of xanthine oxidase or catalase using stroke-prone spontaneously hypertensive rats (SHRSP). In young SHRSP, uric acid content was lower than age-matched Wistar-Kyoto rats (WKY), but in mature SHRSP strongly exposed to oxidative stress uric acid content had risen dramatically. Administration of allopurinol, an inhibitor of xanthine oxidase, caused a marked decrease in uric acid content. In these SHRSP, cerebral injury was much more intense compared to the control group. Administration of aminotriazole, an inhibitor of catalase, did not affect the brain pathol. of SHRSP, in spite of a mild reduction in tissue uric acid content. These results suggest that the xanthine-xanthine oxidase system is not the major source of free radical generation in hypertensive cerebral injury. Moreover, the results also suggest that tissue uric acid may have a key role for the incidence of hypertensive cerebral injury in SHRSP.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 9 OF 102 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:51850 CAPLUS
DOCUMENT NUMBER: 128:113673
TITLE: Possibility of gout complications caused by xanthine oxidase and active oxygen
AUTHOR(S): Matsumoto, Mihuji; Sakano, Shougo
CORPORATE SOURCE: Dep. Blood Transfus., Nagoya City Univ., Nagoya, 467, Japan
SOURCE: Purin, Pirimijin Taisha (1997), 21(2), 171-173
CODEN: PPTAEV; ISSN: 0916-2836
PUBLISHER: Nippon Purin, Pirimijin Taisha Gakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Oxidation and denaturing of blood lipids as well as atherosclerotic changes were detected in gout patients, and atherosclerosis is a factor selecting uric acid controlling medicine in gout. The patients with hypertension, glucose tolerance anomaly and splanchnic

adiposis, being atherosclerotic factors, exhibited high lipid peroxide (LPO) concentration in blood before drug treatment. LPO concentration decreased after treatment in patients with decrease in atherosclerotic factors. The concentration was lower in patients receiving allopurinol (AP) than benzbromarone (BB). The pos. ratio of anti-oxidized and denatured low d. lipoprotein (LDL) antibody was higher in BB-treated patients than AP-treated patients in cases with administration period >1 yr. In BB-treated patients, the LPO concentration increased when hyperuricemia was poorly controlled.

L32 ANSWER 10 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1997018893 EMBASE
TITLE: 6. Gout and other crystal arthropathies.
AUTHOR: McGill N.W.
CORPORATE SOURCE: Dr. N.W. McGill, Royal Prince Alfred Medical Centre, Carillon Avenue, Newtown, NSW 2042, Australia
SOURCE: Medical Journal of Australia, (6 Jan 1997) Vol. 166, No. 1, pp. 33-38.
Refs: 15
ISSN: 0025-729X CODEN: MJAUAJ
COUNTRY: Australia
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
006 Internal Medicine
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Feb 1997
Last Updated on STN: 14 Feb 1997

=> d 11-20 L32 ibib abs

L32 ANSWER 11 OF 102 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 1996:207673 CAPLUS
DOCUMENT NUMBER: 124:313438
TITLE: Potentiation of nitric oxide-mediated vasorelaxation by xanthine oxidase inhibitors
AUTHOR(S): Miyamoto, Yoichi; Akaike, Takaaki; Yoshida, Masaki; Goto, Shingo; Horie, Hidechika; Maeda, Hiroshi
CORPORATE SOURCE: School Medicine, Kumamoto Univ., Kumamoto, 860, Japan
SOURCE: Proceedings of the Society for Experimental Biology and Medicine (1996), 211(4), 366-73
CODEN: PSEBAA; ISSN: 0037-9727
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nitric oxide (NO), now almost synonymous with endothelium-derived relaxing factor (EDRF), reacts with superoxide anion radical (O2-) and forms a potentially toxic mol. species, peroxynitrite (ONOO-). Because xanthine oxidase (XO) seems to be a major O2--producing enzyme in the vascular system, it is important to clarify the mechanism of XO regulation of NO/EDRF. We first characterized the inhibition of XO in vitro by three types of pyrazolopyrimidine derivs. Kinetic studies indicated that 4-amino-6-hydroxypyrazolo[3,4-d]pyrimidine (AHPP) and allopurinol competitively inhibited the conversion of xanthine to uric acid catalyzed by XO, with apparent Ki values of 0.17 ± 0.02 and 0.50 ± 0.03 μM, resp.; alloxanthine inhibited this conversion in a noncompetitive manner with an apparent Ki value of 3.54 ± 1.12 μM. O2- generation in the xanthine/XO system assayed by lucigenin-dependent chemiluminescence was suppressed most strongly by AHPP in a dose-dependent fashion; allopurinol itself appears to reduce the enzyme by transfer of an electron to O2, thus generating O2-. AHPP significantly

augmented EDRF-mediated relaxation of aortic rings from both rabbits and spontaneously hypertensive rats (SHR) in a dose-dependent manner, whereas allopurinol did not affect the relaxation and only marginal potentiation of the vasorelaxation was observed with alloxanthine. Finally, i.v. injection of AHPP (50.4 mg/kg; 100 μmol/300 g rat) reduced the blood pressure of SHR rats to 70% of the initial pressure; this pressure is almost the blood pressure of normal rats. Allopurinol (100 μmol/300 g rat; i.v.) showed transient decrease in blood pressure and moderate reduction of hypertension of SHR (10%) was observed with i.v. injection of alloxanthine (100 μmol/300 g rat). On the basis of these results, it seems that XO regulates EDRF/NO via production of O2-.

L32 ANSWER 12 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1996154453 EMBASE
TITLE: Effects of the specific angiotensin II receptor antagonist losartan on urate homeostasis and intestinal urate transport.
AUTHOR: Hatch M.; Freel R.W.; Shahinfar S.; Vaziri N.D.
CORPORATE SOURCE: Dr. M. Hatch, Department of Medicine, Med Sci I, University of California, Irvine, CA 92717, United States
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (Jan 1996) Vol. 276, No. 1, pp. 187-193.
Refs: 21
ISSN: 0022-3565 CODEN: JPETAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
006 Internal Medicine
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jun 1996
Last Updated on STN: 11 Jun 1996

AB Possible mechanisms for the hypouricemic effects of the angiotensin II receptor antagonist losartan were examined using rats with experimental chronic renal failure (CRF) and control animals. The results show that losartan has a uricosuric effect in rats with normal or decreased renal function. Renal clearance of urate was increased 3-fold in CRF rats and 2- fold in control rats after 7 days of intraperitoneal losartan administration. Although the results show that losartan and its metabolite EXP-3174 alter urate and Cl(-) transport across isolated short-circuited intestine, these agents do not promote urate secretion into the intestinal lumen. Unidirectional urate and Cl(-) fluxes were reduced across normal rat colon and unaltered in the small intestine in the presence of losartan. In CRF rat colon, net secretion of urate and Cl(-) was abolished after losartan addition at 10(-5) M. Transport across the small intestine of CRF rats did not change in the presence of a similar concentration of drug. Losartan treatment of CRF rats before the removal of colonic tissues reversed the basal net secretion of urate to net absorption. These results suggest that the changes in intestinal transport observed in the presence of losartan appear to be mediated via the angiotensin II receptor antagonistic action of this drug. Direct determination of the effects of angiotensin II on urate and Cl(-) transport across colonic tissues from control animals revealed a significant angiotensin II stimulation of urate secretion. These angiotensin II-induced alterations in transport were inhibitable by losartan.

L32 ANSWER 13 OF 102 MEDLINE on STN DUPLICATE 3

ACCESSION NUMBER: 95150005 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7847350
TITLE: Familial hyperuricemic nephropathy.

AUTHOR: Reiter L; Brown M A; Edmonds J
CORPORATE SOURCE: Department of Renal Medicine, St George Hospital, Sydney, Australia.
SOURCE: American journal of kidney diseases : the official journal of the National Kidney Foundation, (1995 Feb) Vol. 25, No. 2, pp. 235-41.
Journal code: 8110075. ISSN: 0272-6386.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 16 Mar 1995
Last Updated on STN: 16 Mar 1995
Entered Medline: 3 Mar 1995

AB This report describes a Polynesian family that had the rare combination of hyperuricemia, precocious gout, hypertension, and renal failure at an early age, with an autosomal dominant inheritance. One family member had renal biopsy evidence of interstitial urate crystal deposition, a surprisingly uncommon observation in such families, and most had decreased fractional excretion of urate, reflecting either decreased secretion or enhanced postsecretory renal reabsorption of uric acid. One patient has had a successful renal transplant. On the basis of these observations, family members of any such index case should be screened for this disorder. Treatment of affected members might include a uricosuric agent and/or allopurinol early in the course of the disorder.

L32 ANSWER 14 OF 102 EMBASE
ACCESSION NUMBER: 1995114382 EMBASE
TITLE: [Should the blood pressure be lowered in patients with gout?].
BLUTDRUCKSENKUNG BEI GICHTPATIENTEN?.
AUTHOR: Kraft K.
CORPORATE SOURCE: Dr. K. Kraft, Medizinische Universitats-Poliklinik, Wilhelmstr. 35-37, 53111 Bonn, Germany
SOURCE: Medizinische Monatsschrift fur Pharmazeuten, (1995) Vol. 18, No. 4, pp. 102.
ISSN: 0342-9601 CODEN: MMPHDB
COUNTRY: Germany
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
033 Orthopedic Surgery
037 Drug Literature Index
LANGUAGE: German
ENTRY DATE: Entered STN: 14 May 1995
Last Updated on STN: 14 May 1995

L32 ANSWER 15 OF 102 EMBASE
ACCESSION NUMBER: 1995214701 EMBASE
TITLE: The influence of allopurinol on renal deterioration in familial nepropathy associated with hyperuricemia (FNAH).
AUTHOR: Miranda M.E.
CORPORATE SOURCE: M.E. Miranda, Division of Internal Medicine, 'La Paz' Hospital, Universidad Autonoma, Madrid, Spain
SOURCE: Advances in Experimental Medicine and Biology, (1995) Vol. 370, pp. 61-64.
ISSN: 0065-2598 CODEN: AEMBAP
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 028 Urology and Nephrology

037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Aug 1995
Last Updated on STN: 3 Aug 1995

L32 ANSWER 16 OF 102 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:664198 CAPLUS
DOCUMENT NUMBER: 123:166783
TITLE: Analysis of 135 cases of primary gouty arthritis
AUTHOR(S): Yi, Wei; Liang, Xueping; Fan, Jiyuan; Zhang, Peng; Jia, Zhiheng
CORPORATE SOURCE: Dep. Endorcinology, Tianjing Med. Univ., Tianjing, 300052, Peop. Rep. China
SOURCE: Tianjin Yiyao (1995), 23(1), 3-6
CODEN: TIYADG; ISSN: 0253-9896
PUBLISHER: Tianjin Yixue Zazhishe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB 135 Of primary gouty arthritis (134 males and 1 female) were complicated with obesity (51.7%), hypertension (43.7%) ischemia heart disease (25.2%), hyperlipemia (37.8%), nephrolithiasis (23.7%), s.c. trophic (18.5%), 15% of them had family history, onset age of gout arthritis ranged from 21 to 74 yr (median 52 yr). After allopurinol therapy serum uric acid decreased from 530.7 ± 99.9 μmol/L (n = 135) to 240.9 ± 66.6 μmol/L (n = 122), p <0.001, fractional clearance (CUA/Ccr) recovered from 7.7 ± 4.7% (n = 96) to 19.4 ± 12.6% (n = 81), p <0.001. 87.6% Patients arthritis were relieved, the results suggested that the deficiency of renal uric acid clearance was involved in the pathogenesis of primary gout.

L32 ANSWER 17 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1994342615 EMBASE
TITLE: Finger pad deposits.
AUTHOR: Eng A.M.; Schmidt K.; Bansal V.
CORPORATE SOURCE: Dr. A.M. Eng, Loyola University Medical Center, Maywood, IL, United States
SOURCE: Archives of Dermatology, (1994) Vol. 130, No. 11, pp. 1433-1434.
ISSN: 0003-987X CODEN: ARDEAC
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
037 Drug Literature Index
038 Adverse Reactions Titles
005 General Pathology and Pathological Anatomy
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Dec 1994
Last Updated on STN: 7 Dec 1994

L32 ANSWER 18 OF 102 MEDLINE on STN
ACCESSION NUMBER: 95390059 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7660979
TITLE: The influence of allopurinol on renal deterioration in familial nepropathy associated with hyperuricemia (FNAH). The Spanish Group for the Study of FNAH.
AUTHOR: Miranda M E
CORPORATE SOURCE: Division of Internal Medicine, La Paz Hospital, Universidad Autonoma, Madrid, Spain.
SOURCE: Advances in experimental medicine and biology, (1994) Vol. 370, pp. 61-4.
Journal code: 0121103. ISSN: 0065-2598.
PUB. COUNTRY: United States

DOCUMENT TYPE: (CLINICAL TRIAL)
(COMPARATIVE STUDY)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 13 Oct 1995
Last Updated on STN: 13 Oct 1995
Entered Medline: 2 Oct 1995

L32 ANSWER 19 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1994189604 EMBASE
TITLE: Recognizing and preventing adverse drug reactions.
AUTHOR: Benjamin D.M.
SOURCE: Drug Therapy, (1994) Vol. 24, No. 6, pp. 52-56.
ISSN: 0001-7094 CODEN: DRTHE2
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20 Jul 1994
Last Updated on STN: 20 Jul 1994
AB A 40-year-old Caucasian man, 5' 10'', 190 pounds, is seen for his annual medical exam. All physical and laboratory findings are within normal limits except that his BP is measured at 170/97, and 172/95 when repeated in the same arm. A decision is made to treat the hypertension, and hydrochlorothiazide 50 mg per day is prescribed. The patient is seen 1 week later and the BP has fallen to 160/92, but the patient complains of lethargy. The patient is crossed-over to a fixed combination product containing hydrochlorothiazide 25 mg and triamterene 50 mg and told to take 2 capsules once a day. Blood is drawn for serum electrolytes, which come back normal. Three months later, the patient's BP has dropped to 142/86, but his uric acid is now 10.2 mg/dL (normal range: 3 to 8 mg/dL) and he reports 'feeling achy.' A decision is made to prescribe allopurinol 100 mg three times daily for the hyperuricemia and ibuprofen 400 mg every 6 hours, prn, for the 'aches and pains.' One week later the patient calls complaining of a rash, feelings of nausea, and reduced urine output over the past 24 hours. What happened and why?

L32 ANSWER 20 OF 102 MEDLINE on STN
ACCESSION NUMBER: 94017223 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8411689
TITLE: Hyperuricemia and atherosclerosis.
AUTHOR: Nishioka K; Iwatani M
CORPORATE SOURCE: Institute of Medical Science, St. Marianna University School of Medicine.
SOURCE: Nippon rinsho. Japanese journal of clinical medicine, (1993 Aug) Vol. 51, No. 8, pp. 2177-81.
Journal code: 0420546. ISSN: 0047-1852.
PUB. COUNTRY: Japan
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199311
ENTRY DATE: Entered STN: 17 Jan 1994
Last Updated on STN: 17 Jan 1994
Entered Medline: 16 Nov 1993

AB To clearly determine whether hyperuricemia participates directly in atherosclerotic disease or not, the prognosis and associated factors were studied, based on data from 104 patients whose serum uric acid had been completely maintained at normal levels with prolonged medication. The mean age at death was 65.8 +/- 10.5 years. The causes of death were as follows: cardiovascular disease (26.9%), cerebral disease (26.2%), malignant neoplasms (26.0%), uremia (7.6%), and miscellaneous disease (18.3%). Serum lipids especially triglycerides, body weight and influenced on the prognosis of the patients FBS. Most common complications were in the cardiovascular disease group; hypertension and hyperlipidemia. These data suggested that the apparent increased incidence of cardiovascular disease in gout rather than renal failure bore a relationship to such complications as hypertension or hypertriglycemia. Hyperuricemia alone may not be an atherosclerotic risk factors. There was no correlation between treatment with allopurinol and probenecid and cardiovascular complications.

=> d L21-30 L32 ibib abs
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in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d 21-30 L32 ibib abs

L32 ANSWER 21 OF 102 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 93151692 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8427538
TITLE: Hereditary nephropathy associated with hyperuricemia and gout.
AUTHOR: Puig J G; Miranda M E; Mateos F A; Picazo M L; Jimenez M L; Calvin T S; Gil A A
CORPORATE SOURCE: Division of Internal Medicine, University Hospital, Madrid, Spain.
SOURCE: Archives of internal medicine, (1993 Feb 8) Vol. 153, No. 3, pp. 357-65.
Journal code: 0372440. ISSN: 0003-9926.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199302
ENTRY DATE: Entered STN: 12 Mar 1993
Last Updated on STN: 12 Mar 1993
Entered Medline: 26 Feb 1993
AB BACKGROUND: The clinical characteristics of hereditary nephropathy associated with hyperuricemia or gout have not been fully described, and the pathogenetic role of increased serum urate concentration is controversial. METHODS: We examined the clinical characteristics of 14 patients and purine metabolism of seven patients, while they were on a purine-restricted diet, in two families with hereditary nephropathy associated with asymptomatic hyperuricemia or gout. Results of plasma and urinary purine measurements were compared with those obtained in 25 patients with gout and renal insufficiency and in 25 normal subjects. Eight subjects in both families were followed up for a mean of 44 months. Allopurinol was given to all patients and enalapril maleate to hypertensive subjects. RESULTS: All patients had some combination of hyperuricemia, gout, renal insufficiency, arterial hypertension, and reduced kidney size. Decreased glomerular filtration rate was

proportional to the decreased renal plasma flow. Renal vascular resistance was markedly increased in the patients with diminished renal plasma flow. All patients with familial nephropathy showed diminished urinary uric acid, hypoxanthine, and xanthine excretion rates. Purine under-excretion was more severe in affected patients with familial nephropathy than in patients with gout and renal insufficiency. Kidney biopsy specimens from three patients with familial nephropathy showed tubulointerstitial lesions and ischemic changes in glomeruli but no uric acid crystals. The kidney uric acid content was normal. Allopurinol treatment normalized serum urate levels, but serum creatinine concentrations increased and creatinine clearance decreased in all patients with familial nephropathy. One patient with gout only at initial evaluation developed renal failure during the follow-up period. CONCLUSIONS: Increased serum urate concentrations in hereditary nephropathy associated with hyperuricemia and gout are due to severe impairment of uric acid excretion. Hyperuricemia does not appear, however, to be of pathogenetic relevance and may be a consequence of a primary disruption of renal hemodynamics.

L32 ANSWER 22 OF 102 EMBASE

ACCESSION NUMBER: 1993086961 EMBASE
TITLE: Allopurinol hypersensitivity syndrome: A review.
AUTHOR: Arellano F.; Sacristan J.A.; Saint-Pierre J.
CORPORATE SOURCE: Dr. J.A. Sacristan, Clinical Research Department, Lilly S.A., Avda. de la Industria, 30, 28100-Alcobendas, Madrid, Spain
SOURCE: Annals of Pharmacotherapy, (1993) Vol. 27, No. 3, pp. 337-343.
ISSN: 1060-0280 CODEN: APHRER
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English; Spanish; Castilian; French
ENTRY DATE: Entered STN: 25 Apr 1993
Last Updated on STN: 25 Apr 1993

AB OBJECTIVE: To review the pathophysiology, pathology, and clinical findings of allopurinol hypersensitivity syndrome (AHS), an infrequent but life- threatening adverse effect of allopurinol therapy. DATA SOURCES: A MEDLINE search (key terms hepatitis, interstitial nephritis, severe hypersensitivity, severe toxicity, vasculitis, toxic epidermal necrolysis, Lyell's syndrome, erythema multiforme, and Stevens-Johnson syndrome) was used to identify cases reported in the literature through the end of 1990. STUDY SELECTION: All cases evaluated met Singer and Wallace's diagnostic criteria for AHS. DATA EXTRACTION: We extracted data from 101 cases of AHS reported in the literature. The following information, when available, was analyzed: (1) patient data (age, gender, medical history), (2) treatment data (daily dosage of allopurinol, duration of treatment, indications, concomitant medications, and (3) adverse-event data. DATA SYNTHESIS: Patients were mostly middle-aged men with hypertension and/or renal failure receiving excessive doses of allopurinol primarily for asymptomatic hyperuricemia. Cutaneous rash and fever were the most common clinical findings. CONCLUSIONS: Although the pathophysiologic pathway leading to the development of AHS is unknown, it probably involves an immunologic mechanism following allopurinol accumulation in patients with poor renal function. Our findings suggest that the accepted diagnostic criteria for AHS may be too broad, and we recommend the application of more restrictive criteria. There is no effective treatment for AHS. The use of allopurinol only for accepted indications and in dosages adjusted for a patient's renal function may be the only

means of minimizing the incidence of AHS.

L32 ANSWER 23 OF 102 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 1994:82078 BIOSIS
DOCUMENT NUMBER: PREV199497095078
TITLE: Randomized prospective study of a nonthiazide diuretic, indapamide, in preventing calcium stone recurrences.
AUTHOR(S): Borghi, Loris [Reprint author]; Meschi, Tiziana; Guerra, Angela; Novarini, Almerico
CORPORATE SOURCE: Inst. Semeiotica Medica, Universita Parma, Via Gramsci 14, 43100 Parma, Italy
SOURCE: Journal of Cardiovascular Pharmacology, (1993) Vol. 22, No. SUPPL. 6, pp. S78-S86. CODEN: JCPCDT. ISSN: 0160-2446.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Feb 1994
Last Updated on STN: 23 Feb 1994

AB We examined the biochemical changes and the efficacy of indapamide in the prevention of calcium stone recurrences. Seventy-five patients with calcium nephrolithiasis and hypercalciuria were randomly assigned to three different therapies: diet and fluid (group A), diet and fluid plus indapamide 2.5 mg/day (group B), and diet and fluid plus indapamide 2.5 mg/day plus allopurinol 300 mg/ day (group C). Before treatment and after 6, 12, 24, and 36 months of therapy, we evaluated blood pressure, serum and urine risk parameters (including relative supersaturations of calcium oxalate, calcium phosphate and uric acid), stone rate, and the proportion of calculi-free patients. During the 3 years of treatment, urinary calcium greatly decreased in groups B and C, dropping to 50% of the pretreatment values; urinary oxalate also significantly declined in group B (- 24%) and group C (- 27%). Relative supersaturations of calcium oxalate and calcium phosphate decreased to the same extent in groups B and C (about one-half of the pretreatment value), and relative supersaturation of uric acid was particularly reduced in group C (-65% of the pretreatment value). The stone rate improved in all three groups (p lt 0.005), but using actuarial analysis in the evaluation of calculi-free patients, indapamide, and indapamide plus allopurinol groups were found to have a significantly more favorable effect than diet and fluid treatment (p lt 0.02), without any difference between the two drug groups. Because indapamide has fewer side effects than thiazide diuretics, we conclude that indapamide could be an interesting alternative to thiazides in the prevention of calcium stones in hypercalciuric patients.

L32 ANSWER 24 OF 102 MEDLINE on STN
ACCESSION NUMBER: 92381924 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1355121
TITLE: The management of hyperuricemia associated with drug treatment of hypertension.
AUTHOR: Nakata T; Iimura O
CORPORATE SOURCE: Second Department of Internal Medicine, Sapporo Medical College.
SOURCE: Nippon rinsho. Japanese journal of clinical medicine, (1992 May) Vol. 50 Suppl, pp. 116-9. Journal code: 0420546. ISSN: 0047-1852.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199209
ENTRY DATE: Entered STN: 18 Oct 1992
Last Updated on STN: 6 Feb 1995
Entered Medline: 25 Sep 1992

L32 ANSWER 25 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1992173094 EMBASE
TITLE: Insidious renal failure.
AUTHOR: Faber M.D.; Narins R.G.
CORPORATE SOURCE: Dr. M.D. Faber, University of Michigan, Medical School, Ann Arbor, MI, United States
SOURCE: Hospital Practice, (1992) Vol. 27, No. 5, pp. 159-164+169+173-176.
ISSN: 8750-2836 CODEN: HOPRBW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 028 Urology and Nephrology
006 Internal Medicine
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jul 1992
Last Updated on STN: 5 Jul 1992

AB A 50-year-old man presented to his primary physician with a one-day history of bilateral flank pain. He denied dysuria, urinary frequency, hesitancy, or gross hematuria. There was no history of urinary tract infection, fever, weight loss, night sweats, arthralgia, or rash. He was afebrile, and his blood pressure was 150/100. Bilateral costovertebral angle tenderness was noted. In addition, the patient had mild, non-nodular prostatic hypertrophy. During a routine physical examination six months earlier, he had complained of low-back pain radiating to the right leg. The pain was attributed to osteoarthritis and treated with propoxyphene. He also had a five-year history of hypertension, well controlled with nadolol, and peptic ulcer disease, treated intermittently with cimetidine. Laboratory studies at that time showed normal electrolytes, BUN 19 mg/dl, creatinine 1.3 mg/dl, albumin 4.9 gm/dl, total protein 7.5 gm/dl, and uric acid 6.8 mg/dl. Urinalysis revealed a specific gravity greater than 1.030, albumin trace, sulfosalicylic acid test 2+, and a negative urinary microscopic examination. Current urinalysis revealed the following: pH 5.5, 2+ albuminuria, 20 WBCs and 3 RBCs per high-power field, and numerous urate crystals. Serum chemistries included BUN 24, creatinine 1.7, uric acid 9.5, and hemoglobin 14.1. An intravenous pyelogram showed a radiolucent calculus obstructing the left ureterovesical junction and several nonobstructing radiolucent stones in the right ureter. His 24-hour urine contained 2,156 mg of uric acid, 74 mg of calcium, and 3.48 gm of protein. The presumptive diagnosis was acute renal insufficiency due to obstructive uropathy, and bilateral basket retrieval of uric acid stones was performed. Allopurinol, 200 mg daily, was prescribed. Serum protein electrophoresis was normal, and serum immunoelectrophoresis showed only hypogammaglobulinemia.

L32 ANSWER 26 OF 102 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 5

ACCESSION NUMBER: 1991:199354 CAPLUS
DOCUMENT NUMBER: 114:199354
TITLE: Nephrotoxicity of allopurinol is enhanced in experimental hypertension
AUTHOR(S): Trachtman, Howard; Valderrama, Elsa; Futterweit, Stephen
CORPORATE SOURCE: Dep. Pediatr., Schneider Child. Hosp., New Hyde Park, NY, 11042, USA
SOURCE: Hypertension (1991), 17(2), 194-202
CODEN: HPRTDN; ISSN: 0194-911X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Hyperuricemia is present in 20-40% of pediatric and adult patients with essential hypertension. This metabolic abnormality may represent an addnl. risk factor for the development of cardiovascular disease. Therefore, the authors performed the following studies to determine

1) whether hyperuricemia is more prevalent in the spontaneously hypertensive rat (SHR) and 2) whether allopurinol treatment has a beneficial effect on the development of hypertension in this strain, based on its capacity to lower the serum uric acid concentration and to act as an antioxidant agent. SHR and control Wistar-Kyoto (WKY) rats were assigned to two groups, one given tap water to drink and the other provided water containing allopurinol (400 mg/L) to furnish an approx. daily dose equal to 100 mg/kg. This treatment was maintained for 15 wk. The serum uric acid levels were similar in untreated SHR and WKY rats (1.85 vs. 1.66 mg/dL). In the control WKY rat strain, allopurinol therapy did not adversely affect weight gain or hematocrit and did not cause an increase in mortality. It resulted in a moderate decrement in kidney function (creatinine clearance: allopurinol-treated group 0.32 vs. control group 0.46 mL/min/100 g body wt, in conjunction with mild-to-moderate tubulointerstitial inflammation (allopurinol-treated group 0.9 vs. control group 0). In contrast, administration of allopurinol to SHR resulted in failure to thrive, marked anemia, severe azotemia (creatinine clearance: allopurinol-treated group 0.04 vs. control group 0.39 mL/min/100 g body weight; $p<0.001$), and severe tubular atrophy and interstitial fibrosis (allopurinol-treated group 2.2 vs. control group 0). These findings indicate that hyperuricemia is not more prevalent in the SHR. Furthermore, allopurinol administration is associated with markedly increased nephrotoxicity characterized by severe tubulointerstitial injury, azotemia, and impaired growth.

L32 ANSWER 27 OF 102 MEDLINE on STN
ACCESSION NUMBER: 90346813 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2384454
TITLE: Clinical profile, therapeutic approach and outcome of gouty arthritis in northern India.
AUTHOR: Kumar A; Singh Y N; Malaviya A N; Chaudhary K; Tripathy S
CORPORATE SOURCE: Department of Medicine, All India Institute of Medical Sciences, New Delhi.
SOURCE: The Journal of the Association of Physicians of India, (1990 Jun) Vol. 38, No. 6, pp. 400-2.
Journal code: 7505585. ISSN: 0004-5772.
PUB. COUNTRY: India
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 26 Oct 1990
Last Updated on STN: 26 Oct 1990
Entered Medline: 20 Sep 1990

AB Thirty patients with gouty arthritis were studied over 3 years. The diagnosis was established with the help of polarised light microscopy. All the patients were males, with a median age of 45 years. They belonged to the middle or upper socio-economic class and were obese (mean body mass index 29.7). Chronic alcoholism, diabetes mellitus and hypertension were present in one patient each. No patient had symptomatic coronary artery disease. Although 6 patients had a history of renal colic, only one had gouty nephropathy with chronic renal failure. Six patients had a positive family history of gout. The disease involved mostly the joints of the lower extremity and podagra was observed in 70% of patients. Eight patients had tophi at various sites. There were 17 'over producers' and 13 'under excretors' of uric acid . The treatment consisted of patient education, symptomatic control with non steroidal anti-inflammatory drugs and/or colchicine and antihyperuricaemic therapy. The overproducers were treated with allopurinol while the under excretors were treated with [corrected] sulfinpyrazone. In general, there was a good response to therapy as indicated by lowering of serum uric acid and the number of painful episodes per year. The overall profile of the

disease appears similar to that seen in the West.

L32 ANSWER 28 OF 102 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1990137206 EMBASE
TITLE: Uric acid metabolism in children.
AUTHOR: Baldree L.A.; Stapleton F.B.
CORPORATE SOURCE: Dr. F.B. Stapleton, Pediatric Research Laboratory, 956 Court Avenue, Memphis, TN 38163, United States
SOURCE: Pediatric Clinics of North America, (1990) Vol. 37, No. 2, pp. 391-418.
ISSN: 0031-3955 CODEN: PCNAA8
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 037 Drug Literature Index
007 Pediatrics and Pediatric Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 1991
Last Updated on STN: 13 Dec 1991
AB The renal excretion of uric acid in children differs quantitatively, and perhaps qualitatively, from that in adult humans. The younger the child, the greater the renal clearance of uric acid and the greater the excretion of uric acid expressed as mg per kg body weight. During infancy, the reduced ability to maximally concentrate the urine may protect against precipitation of uric acid crystals within the kdiney. Conversely, the extremely high urinary uric concentrations places the very small infant at jeopardy during sudden increases in the filtered load of uric acid. Understanding the pharmacologic and physiologic modulators of renal uric acid clearance will allow the pediatrician to minimize the risk of uric acid nephropathy, and to understand the implications of uric acid in the serum or urine in children with fluid and electrolyte disorders. Certainly evaluation of serum and urinary uric acid concentrations is essential in any child with acute renal failure.

L32 ANSWER 29 OF 102 MEDLINE on STN
ACCESSION NUMBER: 90023494 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2801716
TITLE: Evaluation of allopurinol use in patients with gout.
AUTHOR: Zell S C; Carmichael J M
CORPORATE SOURCE: Department of Internal Medicine, Veterans Administration Medical Center, Reno, NV 89520.
SOURCE: American journal of hospital pharmacy, (1989 Sep) Vol. 46, No. 9, pp. 1813-6.
Journal code: 0370474. ISSN: 0002-9289.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198911
ENTRY DATE: Entered STN: 28 Mar 1990
Last Updated on STN: 28 Mar 1990
Entered Medline: 3 Nov 1989
AB The use of long-term allopurinol therapy in patients with gout was evaluated. A pharmacy computer printout was used to identify all outpatients for whom allopurinol had been prescribed during a six-month period in 1985 at a large Veterans Administration medical center. Medical records were reviewed to (1) classify patients as either having or not having definite indications for allopurinol treatment, (2) determine whether physicians had ordered roentgenographic and laboratory tests for presence of monosodium urate crystals,

uric acid excretion, and renal function, and (3) identify gout-associated risk factors and disease entities that could cause hyperuricemia. A pharmacy record of all allopurinol and probenecid prescriptions for the six-month period was obtained, along with cost data. Of the 286 patients who received allopurinol, 32 received the drug for an indication that could not definitely be established as gout. Of the 254 remaining patients, only 45 (17.7%) had a definite indication for allopurinol use as defined by the pharmacy and therapeutics committee. Although pretreatment measurement of serum creatinine was common, only a few patients underwent joint aspiration, a 24-hour urine collection, or roentgenography of affected joints. Large proportions of the patients were found to have gout-associated risk factors. If the 209 patients without definite indications for allopurinol therapy had been treated with probenecid instead of allopurinol, the annual cost savings would have been about $3700. Most of the patients receiving allopurinol for gout could reasonably have been treated with a uricosuric agent such as probenecid at a lower cost. Generally, physicians did not use diagnostic tests optimally before prescribing allopurinol and did not attempt to modify risk factors for gout.

L32 ANSWER 30 OF 102 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 89371795 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2672809
TITLE: Gout in the heart transplant recipient: physiologic puzzle and therapeutic challenge.
AUTHOR: Kahl L E; Thompson M E; Griffith B P
CORPORATE SOURCE: Department of Medicine, University of Pittsburgh School of Medicine, Pennsylvania.
SOURCE: The American journal of medicine, (1989 Sep) Vol. 87, No. 3, pp. 289-94.
Journal code: 0267200. ISSN: 0002-9343.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198910
ENTRY DATE: Entered STN: 9 Mar 1990
Last Updated on STN: 9 Mar 1990
Entered Medline: 6 Oct 1989

AB PURPOSE: Hyperuricemia and gouty arthritis have been associated with cyclosporine use in renal transplant recipients. Patients requiring heart or heart-lung transplantation may have additional risk factors for the development of gout, yet it has not previously been described in this population. We share herein our clinical experience with gouty arthritis in six heart transplant recipients. PATIENTS AND METHODS: During a one-year period, six hospitalized male heart transplant patients were seen in consultation for gouty arthritis. Five were subsequently followed for gout as outpatients; the sixth died within six months. Management included trials of nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, allopurinol, and intra-articular steroid injections, as well as attempts to minimize cyclosporine nephrotoxicity. RESULTS: Three patients had gout in remission at time of transplant surgery, and three others developed gout for the first time two to 45 months after transplantation. Following transplant surgery, both pre-existing and new-onset gout appeared to exhibit an accelerated course, with unusually rapid development of chronic polyarticular disease and tophi in four of the five patients followed for more than six months. Peak serum uric acid levels ranged from 11.0 mg/dL to 16.5 mg/dL. NSAIDs produced reversible renal insufficiency in four patients. Gout-related infections occurred in three patients, one of whom died. CONCLUSION: Acute gouty arthritis may occur in the heart transplant recipient despite concomitant use of immunosuppressive drugs. Cyclosporine, with its attendant hypertension and